

REMARKS:

As the examiner can see, claims 1, 16, 17, 18, 19 and 20 have been amended so as to be directed to a method of triacylglyceride production wherein DNA encoding a glycerol-3-phosphate acyltransferase is introduced into an organism and the triacylglycerides are subsequently harvested from the organism. Support for these amendments may be found throughout the application, for example, at page 10, lines 5-15.

Support for new claim 44 may be found throughout the application, for example, at page 10, lines 10-15.

Support for new claim 45 may be found throughout the application, for example, in Tables 1-6.

Support for new claims 46-51 may be found throughout the application as well as in claims 7-10 as originally filed.

It is noted that in the afore-mentioned Office Action, the election/restriction requirement made by the examiner in the Office Action dated October 3, 2003 was held by the examiner to be proper. It is respectfully requested that the examiner reconsider this position in view of the amendments to the claims discussed above and further in view of the arguments below.

In the Office Action dated October 3, 2003, the Examiner stated that "the inventions listed as Groups I and II, and methods comprising each of SEQ ID NO: 1-10 do not relate to a single general inventive concept under PCT Rule 13.1 because, under PCT Rule 13.2, they lack the same or corresponding special technical features for the following reasons: a method of transforming an organism with DNA encoding a GPAT was known, as taught by the applicant in the specification at page 2 wherein it is disclosed that rapeseed plants were transformed with a GPAT coding sequence, wherein increasing triacylglyceride content in the organism would be inherent in a method comprising the same components and the same steps".

As discussed above, the claims have been amended to be directed to a method of producing triacylglycerides wherein an organism is transformed with GPAT and triacylglycerides are harvested from the organism. Applicant notes

that the amended claims do not include the same steps as the prior art. As discussed below, the prior art does not teach or suggest that GPAT expression will result in increased triacylglyceride production or that a variety of organisms could be transformed with DNA encoding a GPAT protein as a means of increasing the triacylglyceride content of the organism and then harvesting the triacylglycerides from the organism.

It is further noted that the application as filed clearly shows that triacylglyceride levels are increased by transformation with a DNA encoding GPAT in both yeast (Table 3) and plants (Table 5).

It is further noted that SEQ ID NOs 1-3 and 6-8 are DNA and protein sequences respectively of safflower GPAT and variants thereof having GPAT activity. Similarly, SEQ ID NOs 4-5 and 9-10 are DNA and protein sequences of *E. coli* GPAT and a variant thereof having GPAT activity. As can be seen in Tables 3 and 5, all of these sequences resulted in increased oil content compared to control organisms.

In view of this, it is believed that the amendment to the claims overcomes the election/restriction requirement because the invention relates to a single inventive concept: producing increased levels of triglycerides by transforming an organism with GPAT and harvesting the triglycerides from the organism. As discussed herein, this is not taught or suggested in the prior art. Furthermore, it is believed that at least Tables 3 and 5 provide evidence that the nucleotide and protein sequences referred to as SEQ ID NOs 1-10 are equivalent within the invention, as discussed above.

In conclusion, it is noted that as amended, the claims are directed to a method of producing triacylglycerides which is not taught in the prior art. Applicants have shown that the method works in both yeast and plants and that the sequences referred to as SEQ ID NOs 1-10 are equivalent within the invention.

It is therefore believed that claim 1 constitutes a generic claim to a method of producing triacylglycerides comprising transforming any organism with a DNA encoding a GPAT protein and harvesting the triacylglycerides from the organism.

which is not taught or suggested by the prior art, as discussed below. It is further noted that under MPEP 819.01 that the "office may waive election", provided that it does not result in additional work. It is believed (or rather hoped) that searching the additional SEQ ID NOs will not represent significant additional work for the examiner. It is noted that as discussed above, SEQ ID No 1 and 6 are closely related to SEQ ID No 2 and 7 in that SEQ ID No 1 and 6 are intact safflower plastidial GPAT while SEQ ID No 2 and 7 are safflower plastidial GPAT minus the transit peptide. Similarly, SEQ ID 3 and 8 are safflower plastidial GPAT minus the transit peptide plus an ER retention section. As such, the core GPAT sequence of SEQ ID NOs 1, 2, 3, 6, 7 and 8 are essentially identical. Similarly, SEQ ID No 4 and 9 are E. coli GPAT while SEQ ID No 5 and 10 are E. coli GPAT plus an ER retention sequence, meaning that again, the core GPAT sequences for SEQ ID Nos 4, 5, 9 and 10 are essentially identical.

It is further noted that in the amended claims, transforming the organism with a DNA encoding GPAT and harvesting the triglycerides from the organism are separate steps and are not part of the preamble of the claim.

In view of this, it is respectfully requested that the examiner reconsider the restriction/election requirement. In the event that the examiner does not agree, applicant elects claims 1-5, 12, 17, 44, 45, 46 and 47.

Previous claims 1, 2, 3 and 43 were rejected under 35 USC 102(b) as anticipated by Nishizawa.

Nishizawa teaches a method of increasing the unsaturated fatty acid content in membranes particularly in PG which also results in a prominent decrease of saturated PG (column 8, lines 20-26) as a means of improving a plant's tolerance to chilling damage. That is, by changing the unsaturated fatty acid content of the membrane, the composition of the membrane is altered which results in plants which are more resistant to chilling damage. However, Nishizawa does not teach or suggest a method of producing triacylglycerides wherein GPAT is expressed in an organism and triacylglycerides are recovered from that organism. That is, Nishizawa does not teach or suggest increasing total triacylglyceride content and then harvesting the triacylglycerides. It is therefore

believed that the amendments to the claims overcome this objection.

Previous claims 1, 12, 17, 21, 26, 32, 33, 37, 39 and 43 were rejected under 35 USC 102(b) as anticipated by Murata.

It is noted that Murata teaches "altering the saturation of the fatty acids of phosphatidylglycerol by the introduction of an appropriate acyltransferase (page 712-713)" as a means of reducing chilling sensitivity. Murata also states that "overexpression of acyltransferase in the transformed plants does not affect the flux through the two pathways of glycerolipid synthesis but alters the selectivity for the saturated and cis-unsaturated fatty acids at the sn-1 position of phosphatidylglycerol" (paragraph spanning columns on page 711).

Thus, Murata does not teach or suggest that overall triacylglyceride content can be increased by transformation with GPAT or the harvesting of these triglycerides but rather that the membrane composition of an organism can be altered using GPAT. It is therefore believed that the amendments to the claims overcome this objection.

Previous claims 1-5, 12, 17, 21-25, 28, 32, 33, 37, 39 and 43 were rejected under 35 USC 103(a) as unpatentable over Nishizawa in further view of Davies and Bhella.

Specifically, the examiner has stated that Davies teaches transforming Arabidopsis with DNA encoding LPAAT to produce a plant with increased triacylglycerides. On this basis, the examiner has concluded that it would be obvious to effectively substitute LPAAT with GPAT on the basis that GPAT catalyzes the first reaction in triacylglyceride synthesis.

However, applicants respectfully note that Davies does not teach that a DNA encoding LPAAT would produce plants with increased TAG content but rather teaches altering the composition of TAG at a specific position (sn-2) (see US Patent 5,563,058, column 10, lines 12-29).

Regarding Bhella, it is noted that this reference describes the nucleotide sequence of GPAT and also teaches the use of GPAT for changing membrane composition for providing chilling resistance in plants; however, use of GPAT to increase triglyceride content is not taught or suggested by this reference.

Thus, Murata, Nishizawa and Bhella teach that overexpression of GPAT can be used to change membrane composition for example by converting saturated PG to unsaturated PG as a means of providing chilling resistance. As discussed above, this is not applicants' invention which is the use of GPAT to increase overall triglyceride content of an organism and then harvesting the tryglycerides from the organism.

As discussed above, Davies teaches that overexpression of LPAAT alters the composition of a particular fatty acid at a specific position (sn-2) (see US Patent 5,563,058, column 10, lines 12-29). Combining the teachings of Davies and Nishizawa provides two combinations:

(1) examining an LPAAT-overexpressing plant for chilling resistance, that is, determining whether a membrane having a greater proportion of a particular fatty acid at sn-2 imparts chilling resistance on a plant; and

(2) overexpressing GPAT in an organism for producing membranes with higher unsaturated fatty acid composition wherein higher unsaturated content is desirable.

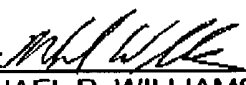
Thus both combinations are directed to methods of changing membrane composition not total triacylglyceride content. Furthermore, neither combination is applicants' invention, which is use of GPAT to produce more triacylglycerides in an organism and recovering the triacylglycerides from the organism.

Furthermore, it is noted that the prior art suggested that other enzymes within the Kennedy pathway, such as DGAT and PAPase were more likely targets for increasing TAG production (see page 1, line 21 to page 2, line 2 of the instant application). Specifically, PAPase and DGAT were believed to be rate-limiting enzymes, not GPAT. It was therefore expected that increasing GPAT activity would not increase activity of the Kennedy pathway overall because the activity of the rate-limiting enzymes had not been modified. That is, based on the prior art, one would anticipate that increasing GPAT activity would provide more substrate for PAPase and/or DGAT but more triacylglycerides would not be produced because PAPase and/or DGAT would still limit the pathway.

In view of the foregoing, further and more favorable consideration is respectfully requested.

Respectfully submitted

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